

***Echocardiographic Assessment of
Pulmonary Artery Parameters Before and After Successful
Percutaneous Transvenous mitral Commissurotomy in
Rheumatic Mitral Stenosis***

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CERTIFICATE

This is to certify that the dissertation titled “*Echocardiographic Assessment of Pulmonary Artery Parameters Before and After Successful Percutaneous Transvenous mitral Commissurotomy in Rheumatic Mitral Stenosis*” is the bonafide original work of Dr. **P.Ramachandran**, in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2014. The period of post-graduate study and training was from August 2011 to July 2014.

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DECLARATION

I, **Dr.P.RAMACHANDRAN**, solemnly declare that this dissertation entitled, *Echocardiographic Assessment of Pulmonary Artery Parameters Before and After Successful Percutaneous Transvenous mitral Commissurotomy in Rheumatic Mitral Stenosis*” is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2011 – 2014 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor M.S.Ravi M.D.D.M. This dissertation is submitted to The Tamilnadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology.**

Place:

SIGNATURE OF THE CANDIDATE

Date

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CONTENTS

	PAGE NO
1. INTRODUCTION	1
2. AIMS AND OBJECTIVES	4
3. REVIEW OF LITERATURE	5
4. MATERIALS AND METHODS	26
5. RESULTS	35
6. DISCUSSION	47
7. CONCLUSION	56
8. LIMITATION OF STUDY	57
9. APPENDIX	
a. Bibliography	
b. Acronyms	
c. Proforma	
d. Master chart	
e. Ethical committee approval order	
f. Plagiarism report	
g. Patient consent form	

INTRODUCTION

INTRODUCTION

The Most common cause of Mitral stenosis in developing countries like india is rheumatic fever,^[19] with rheumatic manifestations present in almost 99% of mitral stenosis patients. 25% of RHD patients have isolated MS, and 40% have combined MS and MR.

Involvement of multivalve is seen in 38% patients, in about 35% have aortic valve and the tricuspid valve in about 6%. Rarely pulmonic valve is affected.

In acute rheumatic fever, there is inflammation and edema of the leaflets, with small fibrin-platelet thrombi along the leaflet contact zones. Subsequently patients develop scarring leads to the characteristic valve deformity, with obliteration of the normal leaflet architecture by fibrosis, neovascularization and increased collagen and tissue cellularity.

Almost Two thirds patients with rheumatic MS are female. The onset of rheumatic fever and clinical evidence of MV obstruction is variable, ranging from a few years to more than 20 years.^{[. [20]}

In developing countries- like india mitral stenosis progresses much more rapidly, because of more severe or repeated streptococcal infections,

genetic influences, or economic conditions, and may lead to symptoms in the late teens and early twenties.

Asymptomatic or minimally symptomatic patients 10 years Survival is >80% without treatment.

Once patients develop symptoms (Functional Class III/IV) , survival without treatment predictably worsens and has been around at 0–15% over the subsequent 10 years.

Mortality rate of patients with mitral stenosis without treatment is attributable to progressive heart failure in 60– 70% of patients, systemic embolism in 20–30%, pulmonary embolism in 10%, and infection in 1–5%.

Initial presentation of patients with mild and moderate mitral stenosis (MVO 1.5 to 2.0 cm²) may be due to exercise , fever, pregnancy, emotional upset or atrial fibrillation, especially with a rapid ventricular response which precipitate the symptoms.

In these patients Percutaneous Transvenous mitral Commissurotomy helpful to avoid early surgery.

PTMC is the procedure of choice for the MS patients so that surgical treatment is now reserved for patients who require treatment and valve morphology not suitable for a percutaneous procedure.

Various invasive catheterisation studies confirmed reduction in pulmonary artery parameters After PTMC. In our study we used non invasive echocardiographic parameters to confirm the reduction in pulmonary artery parameters and how much extent theses parameters decreased after the procedure.

We also studied, is there reduction of pulmonary artery parameters after the onset of mitral regurgitation as a complication of PTMC procedure.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

To analyze Pulmonary Artery Parameters before and after Successful PTMC, in Rheumatic Mitral Stenosis patients by Echocardiography.

INCLUSION CRITERIA

Patients with isolated moderate and severe rheumatic MS who are candidates for intervention in the form of NYHA class \geq II, planimetry MVA $<1.5 \text{ cm}^2$ and echocardiographic favorability for PTMC in the form of mitral regurgitation \leq II/IV and suitable valve morphology were included.

All patients undergoing successful PTMC in Rheumatic Mitral stenosis with suitable criteria between July 2013 and March 2014 in the department of Cardiology, RGGGH, Chennai were included in the study.

EXCLUSION CRITERIA

Patients with Systemic hypertension, Diabetes mellitus, More than mild mitral or aortic regurgitation and/or aortic stenosis or with any other heart muscle diseases were excluded.

Unsuccessful PTMC cases and patients with RV dysfunction as well as

Patients who were unwilling to give consent for the study were excluded.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Mitral stenosis

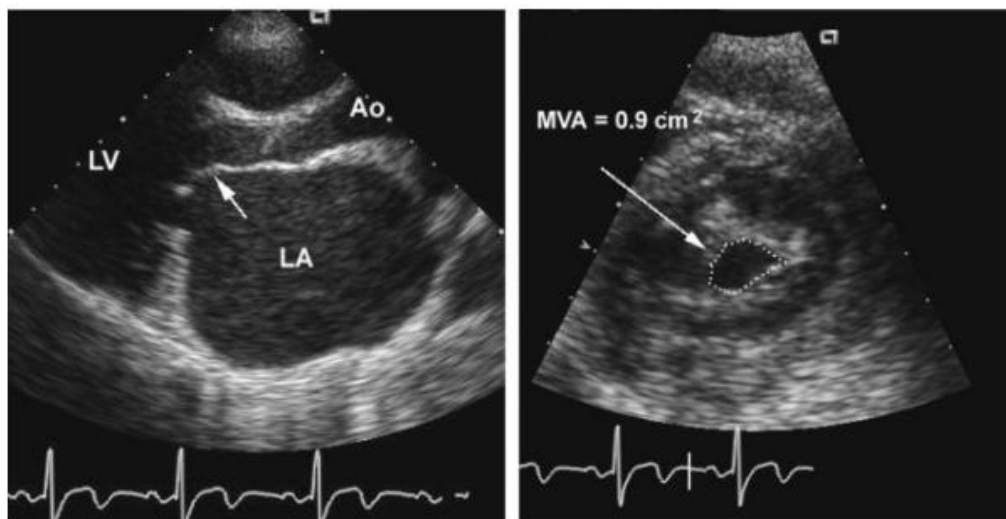
Mitral stenosis is characterized by inflow obstruction of left ventricle at the level of mitral valve due to abnormality of the mitral valve apparatus. Rheumatic fever is the most common cause of mitral stenosis . Other rare etiologies include congenital mitral stenosis, rheumatoid arthritis , carcinoid disease, SLE , Fabry disease ,mucopolysaccharidoses of the Hunter-Hurler phenotype, methysergide therapy and Whipple disease.

Diagnostic features of rheumatic mitral stenosis is leaflet thickening at the edges, fusion of the commissures, and chordal shortening .

With acute rheumatic fever, there is inflammation and edema of the leaflets, with small fibrin-platelet thrombi along the leaflet contact zones. Subsequent occurrence of scarring leads to the characteristic valve deformity, with obliteration of the normal architecture of leaflet by fibrosis, neovascularization and increased collagen and tissue cellularity.

In initial stages of the disease, the relatively flexible leaflets snap open in diastole into a curved shape because of restriction of motion at the

leaflet tips. This diastolic doming is more evident in the motion of the anterior leaflet. Becomes less prominent as the affected leaflets become more fibrotic and calcified.



{Parasternal long-axis **(left)** and short-axis **(right)** 2D echocardiographic views showing the characteristic findings in rheumatic MS. Note the commissural fusion that results in doming of the leaflets in the Parasternal long-axis view and in a decrease in the width of the MVO in the short-axis view}

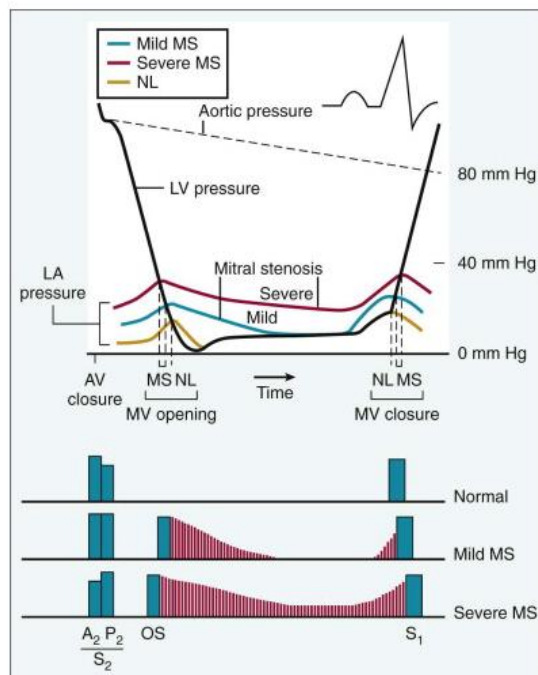
The symmetrical fusion of the commissures results in a central small oval orifice in diastole. In autopsy specimens is shaped like a fish mouth or buttonhole because the anterior leaflet is not in the physiological open position.

Recurrent infection as an important determinant in disease progression. Restenosis after mitral valvuloplasty is caused by fibrosis and leaflet thickening rather than recurrent commissural fusion.

Pathophysiology

The useful descriptor of the severity of mitral valve obstruction is mitral valve orifice area in diastole. In normal adults, mitral valve orifice area is 4 to 6 cm². When the orifice is reduced to approximately 2 cm², which is considered to represent mild MS, blood can flow from the left atrium to the left ventricle only if propelled by a small, although abnormal, pressure gradient. When the mitral valve opening is reduced to 1 cm², which is considered to represent severe MS,^[12] a left atrioventricular pressure gradient of approximately 20 mm Hg (and therefore, in the presence of a normal LV diastolic pressure, a mean left atrial pressure >25 mm Hg) is required to maintain normal cardiac output at rest.

The elevated left atrial pressure raises pulmonary venous and capillary pressures, resulting in exertional dyspnea. The first bouts of breathlessness in patients with MS are usually precipitated by tachycardia resulting from exercise, pregnancy, hyperthyroidism, anemia, infection, or AF.



All these conditions (1) increase the rate of blood flow across the mitral orifice, resulting in further elevation of the left atrial pressure, and (2) decrease the diastolic filling time, resulting in a reduction in forward cardiac output. Because diastole shortens proportionately more than systole as heart rate increases, the time available for mitral valve flow is reduced at higher heart rates. Therefore, at any given stroke volume, increase heart rate results in a higher instantaneous volume flow rate and higher transmitral pressure gradient, which elevates left atrial pressures further. This higher transmitral gradient, often in combination with inadequate ventricular filling (because of the shortened diastolic filling time), explains the sudden occurrence of dyspnea and pulmonary congestion and edema in previously asymptomatic patients with MS who develop AF with a rapid ventricular rate.

Elevated left atrial pressure results in pulmonary venous and artery hypertension, with secondary effects on the pulmonary vasculature and right heart. In addition, left atrial enlargement and stasis of blood flow is associated with an increased risk of thrombus formation particularly in appendages and systemic embolism. Typically, the left ventricle is usually normal, unless there is coexisting MR, with the primary abnormalities of the left ventricle being a small underfilled chamber and paradoxical septal motion caused by RV enlargement and dysfunction.

Mitral valve area is estimated by direct planimetry from two-dimensional short-axis images and calculated by the Doppler pressure half-time method. The transmitral gradient is calculated with use of continuous wave Doppler and any coexisting Mitral regurgitation is quantitated. Evaluation of the morphological status of the mitral valve is important that useful for predicting the hemodynamic status and outcome of PTMC.

Hemodynamic Progression

Overall rate of progression was a decrease in valve area of $0.09 \text{ cm}^2/\text{yr}$. Approximately one third of patients with mitral stenosis showed rapid progression, defined as a decrease in valve area greater than $0.1 \text{ cm}^2/\text{yr}$.

Clinical Outcomes

In the presurgical era MS patients with symptoms have a poor clinical outcome, with 5-year survival rates of 62% among patients with Mitral stenosis in NYHA Class III but only 15% among those in Class IV. In the surgical era not operated patients still reported a 5-year survival rate of 44% in patients with symptomatic MS who refused operation.

Patients who underwent percutaneous or surgical relief of valve obstruction on the current guidelines based therapy have great clinical outcomes. However, longevity is reduced compared with that expected for age, mainly because of complications of the disease process such as AF, systemic embolism, pulmonary hypertension and side effects of therapy such as prosthetic valves, anticoagulation.

Echocardiography

MVO estimation by planimetry (Valve area (cm²))

Mild MS >1.5

Mod MS 1.0-1.5

Severe MS <1.0

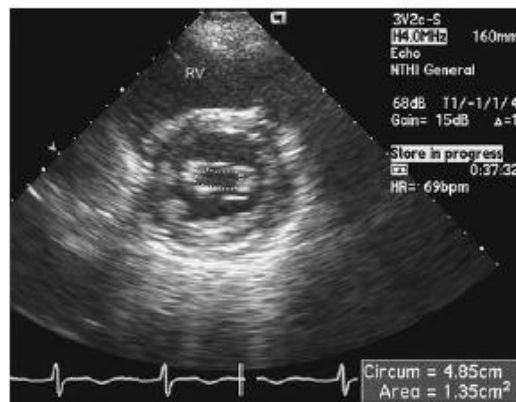


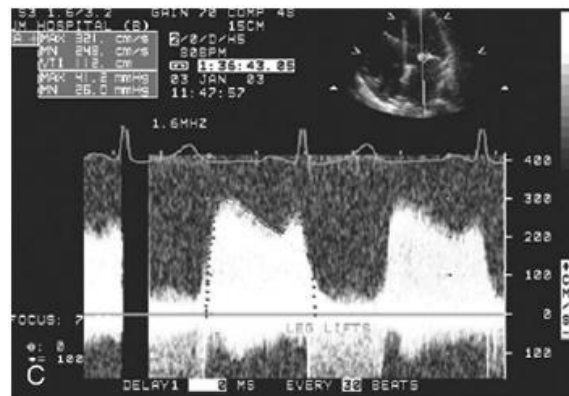
Image is recorded at the level of tip of mitral valve.

Mitral valve pressure gradient Estimation(Mean gradient (mm Hg))

Mild MS <5

Mod MS 5-10

Severe MS >10



Pulmonary Artery Systolic pressure estimation

PASP estimated from TR velocity using continuous wave doppler

$PASP = 4 (Tricuspid\ regurgitant\ velocity)^2 + RA\ pressure]$.

PA Diastolic Pressure

PADP can be evaluated from the velocity of the end-diastolic PR jet using the modified Bernoulli equation with CW doppler:

[PADP = 4 (end-diastolic pulmonary regurgitant velocity)² + RA pressure].

Mean PA Pressure

Mean Pulmonary Artery pressure = 1/3(Systolic PAP)+ 2/3(PADP)

Mean Pulmonary Artery pressure = 79 - (0.45 x AT)

Patients with Acceleration time < 120 ms, the formula for

Mean PAP is= 90 - (0.62 x AT)

Mean PAP can also be estimated as 4 x(early PR velocity)²

+ estimated RA pressure.

Pulmonary Vascular Resistance

PVR = TR Velocity x 10/ RVOT VTI + 0.16

Pulmonary Artery Diameter

Pulmonary artery dimension measured between Bifurcation point and Pulmonary valve



Echocardiographic scoring system divides patients into three groups

Group 1- those with a pliable, noncalcified anterior leaflet and little chordal disease

Group 2- those with a pliable, noncalcified anterior leaflet but with chordal thickening and shortening (<10 mm long;)

Group 3- those with fluoroscopic evidence of calcification of any extent of the valve apparatus.^[13]

Event-free survival at 3 years is highest for group 1 (89%) compared with group 2 (78%) or group 3 (65%).^[12,13]

In our study we used Wilkins echocardiographic scoring system.

Wilkins Score

GRADE	MOBILITY	SUBVALVULAR THICKENING	THICKENING	CALCIFICATION
1	Highly mobile valve with only leaflet tips restricted	Minimal thickening just below the mitral leaflets	Leaflets nearly normal in thickness (4-5 mm)	A single area of increased echo brightness
2	Leaflet mid and base portions have normal mobility	Thickening of chordal structures extending up to one third of the chordal length	Mid leaflets normal, considerable thickening of margins (5-8 mm)	Scattered areas of brightness confined to leaflet margins
3	Valve continues to move forward in diastole, mainly from the base	Thickening extending to the distal third of the chords	Thickening extending through the entire leaflet (5-8 mm)	Brightness extending into the midportion of the leaflets
4	No or minimal forward movement of the leaflets in diastole	Extensive thickening and shortening of all chordal structures extending down to the papillary muscles	Considerable thickening of all leaflet tissue (>8-10 mm)	Extensive brightness throughout much of the leaflet tissue

Excellent immediate and long-term results with score of 8 or less, but scores higher than 8 are associated with less impressive results including the complication of new onset of MR.

Commissural calcification also is a predictor of poor outcomes

Estimation of the right atrial pressure

For Easy estimation and uniformity of reporting, specific values of Right Atrial pressure, rather than ranges, used in the determination of Systolic PAP.

IVC diameter less than 2.1 cm that collapses >50% with a deep inspiration suggests normal Right Atrial pressure of 3 mm Hg (range, 0-

5 mm Hg), whereas Inferior vena cava diameter > 2.1 cm that collapses $< 50\%$ with a deep inspiration suggests high Right Atrial pressure of 15 mm Hg (range, 10-20 mm Hg).

In situations in which Inferior Vena Cava diameter and collapse do not fit this criteria, an intermediate value of 8 mm Hg (range, 5-10 mm Hg) may be used or, preferably, other indices of Right Atrial pressure should be included to upgrade or downgrade to the normal or high values of Right Atrial pressure.

Intermediate (8mmHg) RA pressures may be downgraded to normal (3mmHg) if no secondary indicators of elevated RA pressure are present, upgraded to high if minimal collapse with deep inspiration ($<35\%$) and secondary indices of elevated RA pressure are present, or left at 8 mm Hg if uncertain.

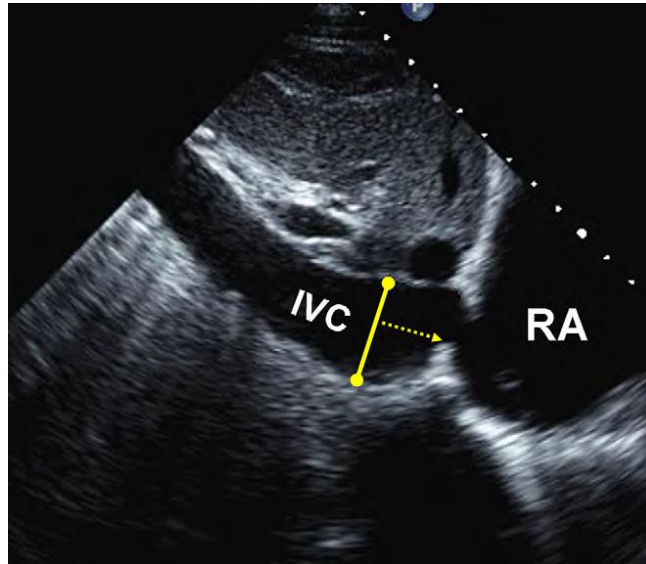
In patients who are not able to adequately perform a deep inspiration , an IVC that collapses $< 20\%$ with quiet inspiration suggests elevated Right Atrial pressure.

Estimation of RAP on the basis of IVC diameter and collapse

Variable	Normal (0-5 [3] mm Hg)	Intermediate (5-10 [8] mm Hg)	High (15 mm Hg)
IVC diameter	<2.1 cm	<2.1 cm >2.1 cm <50% >50%	>2.1 cm
Collapse with sniff	>50%		<50%
Secondary indicators of elevated RAP			<ul style="list-style-type: none"> _ Restrictive filling _ Tricuspid E/E0 > 6 _ Predominant Diastolic flow in hepatic veins (Filling fraction in systole< 55%)

Measurement of the IVC.

The diameter is estimated perpendicular to the long axis of the Inferior Vena Cava at end-expiration, just proximal to the junction of the hepatic veins that present roughly 0.5 to 3.0 cm proximal to the ostium of the RA. During Deep inspiration, IVC diameter is measured.



Management strategy for patients with mitral stenosis

Medical treatment

Prevention of recurrent rheumatic fever.

Prevention and treatment of complications of MS

Medical therapy with oral diuretics and the restriction of sodium intake may improve symptoms .

Beta-blocking agents and calcium antagonists may increase exercise capacity by reducing heart rate in patients with sinus rhythm, especially in patients with AF.

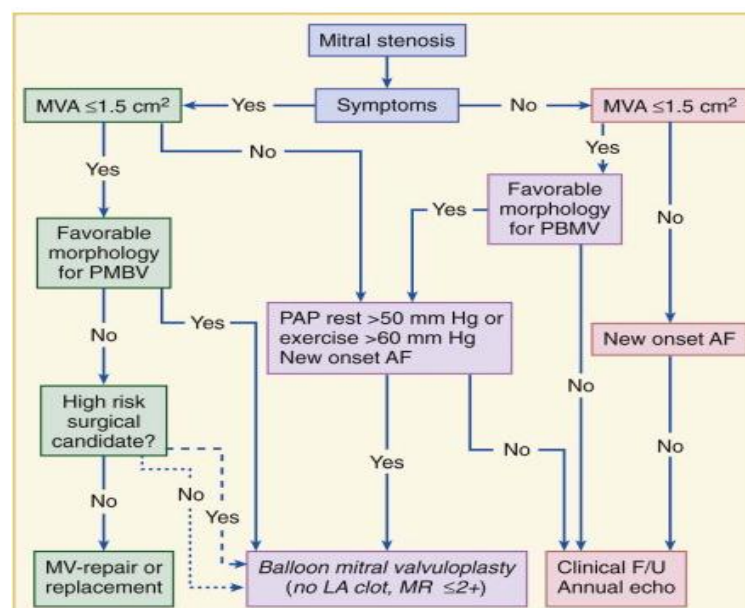
Advised to avoid occupations requiring strenuous exertion.

Anemia and infections should be treated promptly.

Anticoagulant therapy is indicated for prevention of systemic embolism in MS patients with AF.

Monitoring disease progression to allow intervention at the optimal time point

Algorithm for Intervention



Interventional treatment

Percutaneous transvenous mitral commissurotomy

Percutaneous Balloon Mitral Valvotomy

Patients with mild and moderate MS who are asymptomatic commonly remain so for years. But, severe or symptomatic MS patients have poor

long-term results if the stenosis is not relieved with interventional treatment .

Percutaneous Balloon Mitral Valvotomy is the choice of procedure for the treatment of Mitral Stenosis so that surgical treatment is now reserved for patients who require treatment and are not candidates for a percutaneous intervention procedure.^[13]

BMV is recommended for symptomatic patients with moderate to severe MS and with favorable valve morphology, no or mild MR, and no evidence of left atrial thrombus . Even mild symptomatic patients, such as a subtle decrease in exercise tolerance, are an indication for intervention because the procedure relieves symptoms and improves long-term outcome with a low procedural risk. In addition, BMV is recommended for asymptomatic patients with moderate to severe MS when mitral valve obstruction has resulted in pulmonary artery hypertension with a pulmonary systolic pressure greater than 50 mm Hg at rest or 60 mm Hg with exercise.

Even when valve morphology is not suitable, BMV is reasonable for symptomatic patients who are at higher risk for surgical treatment, including patients with restenosis after a previous Balloon Mitral

Valvotomy or previous commissurotomy who are unsuitable for surgery because of very high risk.

BMV also indicated in very old patients, patients with associated severe IHD, patients in whom MS is complicated by renal, pulmonary, or neoplastic disease, women of reproductive age in whom MV replacement is undesirable, and pregnant women with Mitral Stenosis. Percutaneous BMV may be considered for patients with moderate to severe MS and new-onset Atrial Fibrillation.

PTMC

Antegrade approach

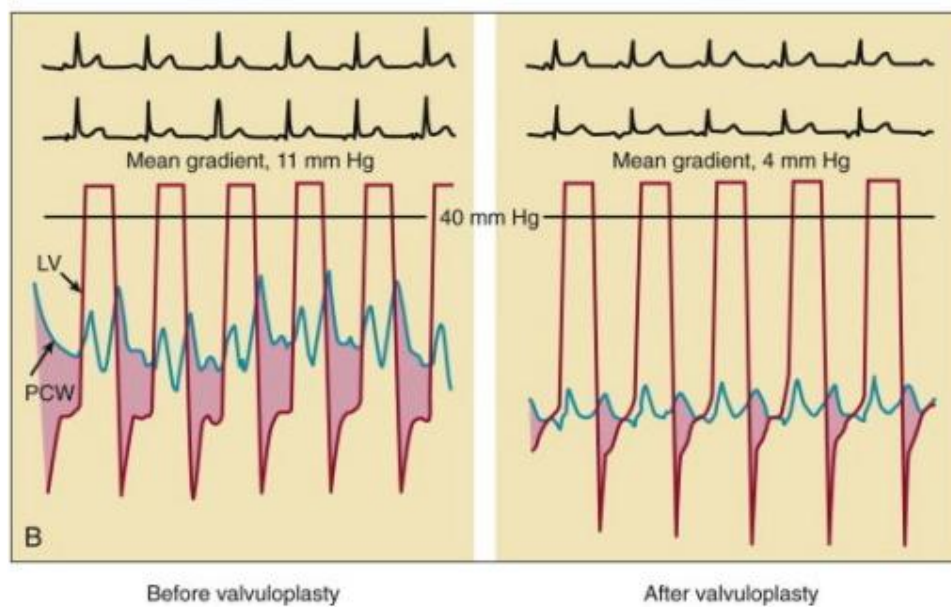
With Inoue balloon

With side by side two smaller (15- to 20-mm) balloons

With Accura balloon

Retrograde approach

Through Aortic valve



Improvement in valvular function due to Commissural separation and fracture of nodular calcium. In several studies reported with decrease of the mitral Mean pressure gradient from 18 mmHg to 6 mm Hg, average 20% increase in cardiac output, and an average increase of the calculated MVO, from 1 to 2 cm². [13-14]

In younger patients results are impressive when severe valvular thickening or calcification is absent. Elevated pulmonary vascular resistance declines rapidly. Mortality rate has ranged from 1% to 2%. Complications include cardiac perforation and cerebral emboli, each in roughly 1% of patients, and the development of Mitral regurgitation severe enough to require surgery in another 2%.

Rigid thickened valves with extensive subvalvular fibrosis and calcification lead to suboptimal results.

A prospective randomized trial in which patients with severe MS ,similar clinical outcomes with BMV and the open surgical technique that were superior to the results of the closed surgical valvotomy. After 7 years of procedure , mitral valve area was equivalent in the BMV and open surgical groups, both significantly greater than in the closed valvotomy group.

Surgical treatment

Closed Mitral commissurotomy

Open mitral vlvotomy

Mitral Valve Replacement

Surgical intervention for mitral stenosis is recommended for patients with severe Mitral Stenosis and symptoms (NYHA Class III or IV) when PTMC is not available, PTMC is contraindicated because of persistent left atrial clot or moderate to severe MR, or when the calcified valve and surgical risk is acceptable.^[15] The most preferred surgical approach is valve repair (open mitra valvotomy, with or without additional procedures) whenever possible.

APPROACH	ADVANTAGES	DISADVANTAGES
Closed surgical valvotomy	<p>Inexpensive</p> <p>Relatively simple</p> <p>Good results in selected patients</p> <p>Good long-term outcome</p>	<p>No direct visualization of valve, feasible with flexible, noncalcified valves.</p> <p>Contraindicated if MR > 2+</p> <p>Surgical procedure</p>
Open surgical valvotomy	<p>Visualization of valve allows directed valvotomy</p> <p>Subsequent annuloplasty for MR is feasible</p>	<p>Best results with flexible, noncalcified valves</p> <p>Surgical procedure.</p>
Valve replacement	<p>Feasible in all patients regardless of any extent of valve calcification or severity of MR</p>	<p>Surgical procedure</p> <p>Effect of loss of annular-papillary muscle continuity on LV function</p> <p>Prosthetic valve</p> <p>Chronic anticoagulation</p>
Balloon mitral valvotomy	<p>Percutaneous approach</p> <p>Local anesthesia</p> <p>Good hemodynamic results in selected patients</p> <p>Good long-term outcome</p>	<p>No direct visualization of valve</p> <p>Only feasible with flexible noncalcified valves</p> <p>Contraindicated if MR > 2+</p>

Review of studies

Pulmonary arterial systolic pressure

Abdenasser Drighil¹ et al studied Immediate impact pulmonary arterial systolic pressure after successful percutaneous mitral valve commissurotomy. This study showed reduction of PASP from 46.4 ± 32.1 to 29.1 ± 13.4 mmHg after PTMC. In these patients MVO increased from 0.91 ± 0.29 to 1.86 ± 0.43 . Mean transmitral gradient (mmHg) decreased from 16.4 ± 8 to 8.5 ± 1.5 mmHg.

Ahmad Noor⁴ et al studied Determinants of Decrease in Pulmonary Hypertension Following PTMC. In this study PASP decreased from 60.12 ± 17.9 mmHg to 33.4 ± 11.9 (mmHg) P value $< 0.01^*$. In this study, PASP decreased by 29%.

Fawzy et al² study showed Systolic pulmonary artery pressure reduction of PASP from 48.3 ± 17.4 to 40 ± 13.7 mmHg after PTMC.

Pulmonary artery mean pressure

Kamal Saad Mansour et al³ study showed Mean PAP 38.12 ± 8.43 mmHg before PTMC that decreased immediately after PTMC to mean value of 32.78 ± 7.89 mmHg.

Pulmonary vascular resistance

Kamal Saad Mansour et al³ study showed ,PVR before PTMC mean value of 2.25 ± 0.66 wu decreased to mean value of 1.76 ± 0.52 wu after PTMC.

Lung B, Vahanian A⁵: *Echocardiography in the patient undergoing catheter BMV*: et al study showed reduction in Pulmonary vascular resistance.

Pulmonary artery acceleration time

*Kamal Saad Mansour*³ et al study showed Pulmonary artery acceleration time before PTMC with mean value of 83.78 ± 16.87 m.sec. that immediately after PTMC increased to mean value of 94.42 ± 15.76 m.sec.

MATERIALS AND METHODS

MATERIALS AND METHODS

Study Population

A total of 35 patients were enrolled for the study from the patients who presented to Cardiology Department Govt General Hospital from the period 1st July 2013 to March 31st 2014.

5 patients were excluded as per exclusion criteria.

The remaining 30 patients, who satisfied all the inclusion criteria were selected for the study . Written consent was obtained from all patients participating in the study.

The control group consisted of 10 age-matched healthy persons who ranged in age from 18 to 42 years (mean age 28 years). None had ECG or echocardiographic evidence of structural or functional cardiovascular disease.

Patients did not have systemic hypertension, diabetes mellitus, more than mild aortic regurgitation and/or aortic stenosis nor New York Heart Association functional class IV symptoms.

Indications for PTMC were New York Heart class \geq II, \leq IV, planimetered MVA $< 1.5 \text{ cm}^2$, mitral regurgitation $\leq 2+$, suitable valve

morphology by Wilkins score, and absence of concomitant cardiovascular disease requiring surgical correction.

All patients were subjected to a detailed Echocardiographic examination as per proforma.

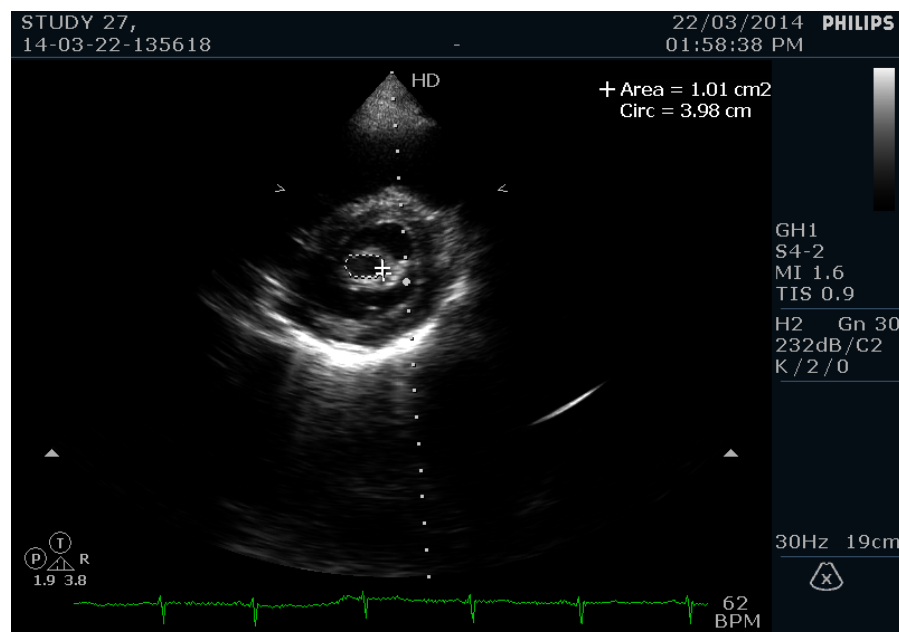
Echocardiogram was done using Philips HD7XE Echocardiographic machine.

Echocardiographic measurements

The following echocardiographic parameters were done.

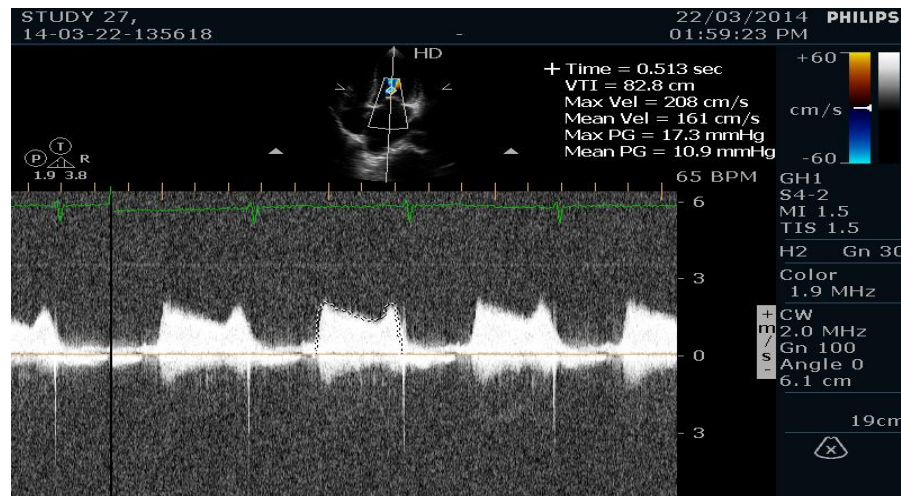
MVO estimation

MVO estimation was done by planimetry using two dimensional short axis view at the level of mitral valve tips.



Mitral valve pressure gradient estimation

Mitral valve pressure gradient estimation was done with continuous doppler.



Mitral regurgitation estimation

Mitral regurgitation estimation by PISA method was done before and after PTMC.¹⁶

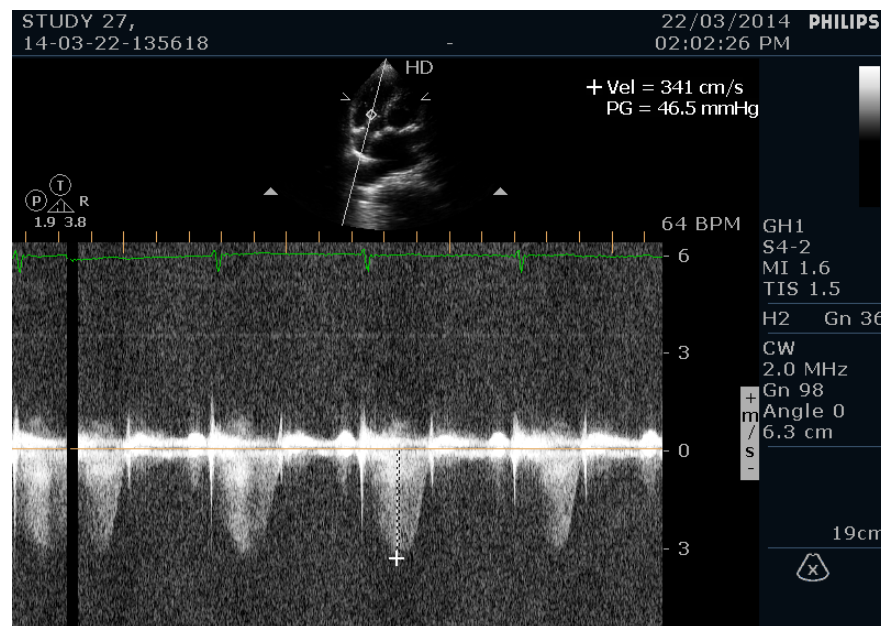
MR Severity¹⁶

Grades	Grade 1	Grade 2	Grade 3	Grade 4
Severity	Mild	Moderate		Severe
Regurgitation volume	< 30 ml	30 to 44 ml	45 to 59 ml	> 60 ml

Pulmonary Artery Systolic pressure¹¹

PASP estimation was done with Tricuspid regurgitant velocity with following formula

$$\text{PASP} = 4 (\text{Tricuspid regurgitant velocity})^2 + \text{RA pressure}].$$



Mean Pulmonary Artery Pressure¹¹

Mean Pulmonary artery Pressure is derived from Pulmonary artery acceleration time with the following formula.

$$\text{Mean PA pressure} = 79 - (0.45 \times \text{AT})$$

Patients with Acceleration time < 120 ms, the formula for Mean Pulmonary Artery pressure is= 90 - (0.62 x AT)

Pulmonary Artery Diastolic pressure¹¹

Pulmonary Artery Diastolic pressure is estimated from PASP and Mean Pulmonary artery Pressure with the following formula.

$$PADP = 3 \text{Mean PAP} - \text{PASP} / 2$$

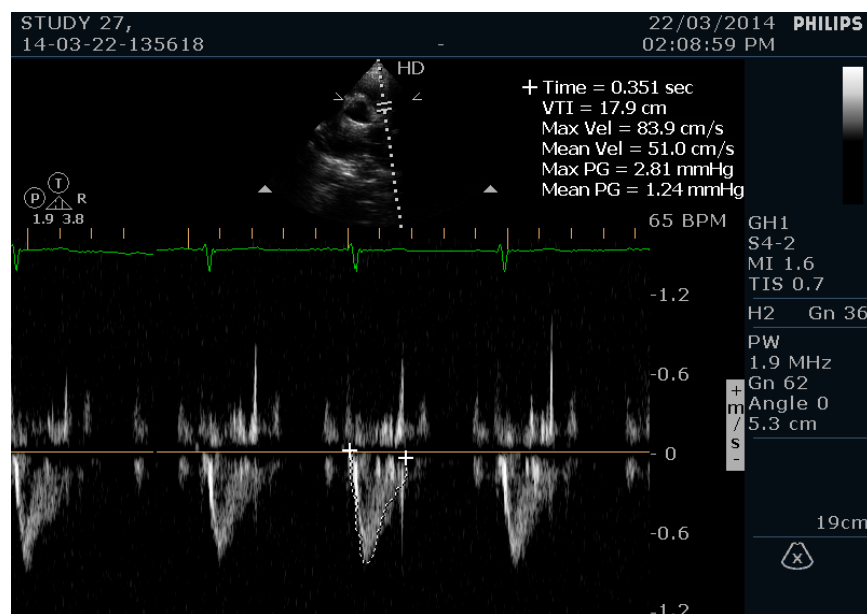
This formula derived from

$$\text{Mean PAP} = 1/3 \text{ rd PASP} + 2/3 \text{ rd PADP}$$

Pulmonary Vascular Resistance¹¹

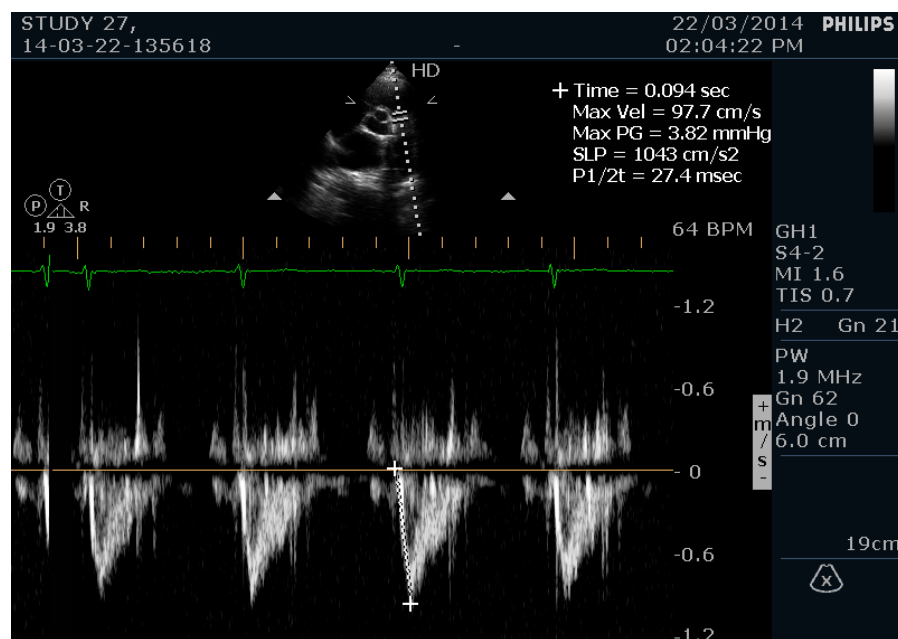
Pulmonary Vascular Resistance is calculated from the following formula

$$PVR = \text{TR Velocity} \times 10 / \text{RVOT VTI} + 0.16$$



Pulmonary artery acceleration time^{II}

Pulmonary artery acceleration time is measured with pulse wave Doppler. Pulse wave doppler cursor is positioned just beyond pulmonary valve tips. The time taken from onset of systole to peak velocity is measured.



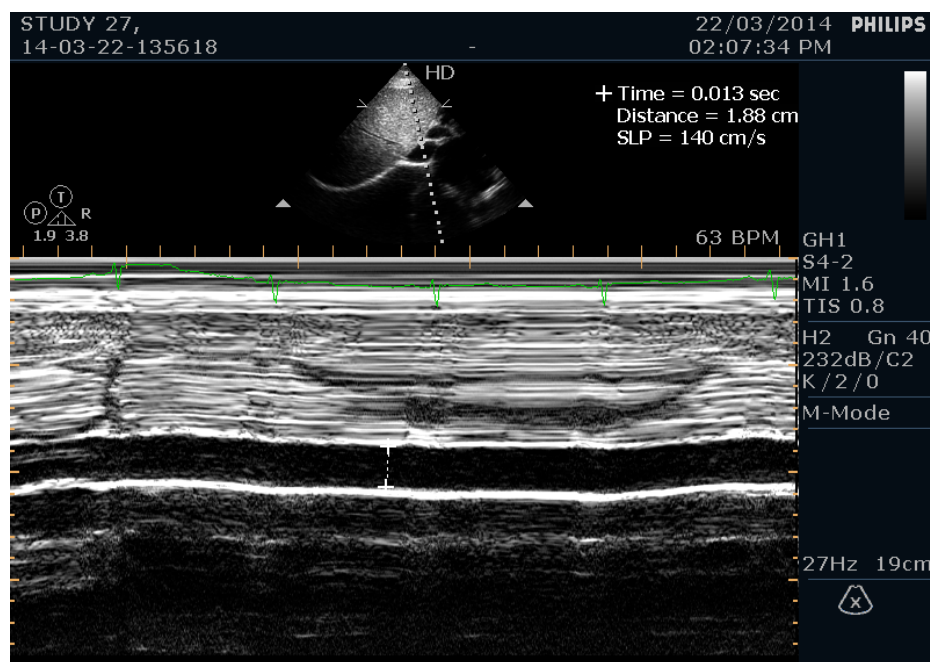
Right atrial pressure estimation^{II}

RAP is estimated from IVC.

IVC diameter less than 2.1 cm that collapses >50% with a deep inspiration indicates normal RA pressure of 3 mm Hg (range, 0-5 mm Hg), whereas Inferior Vena Cava diameter > 2.1 cm that collapses <

50% with a deep inspiration indicates high RA pressure of 15 mm Hg (range; 10-20 mm Hg).

In situations in which Inferior Vena Cava diameter and collapse do not fit this criteria, an intermediate value of 8 mm Hg (range; 5-10 mm Hg) is used.

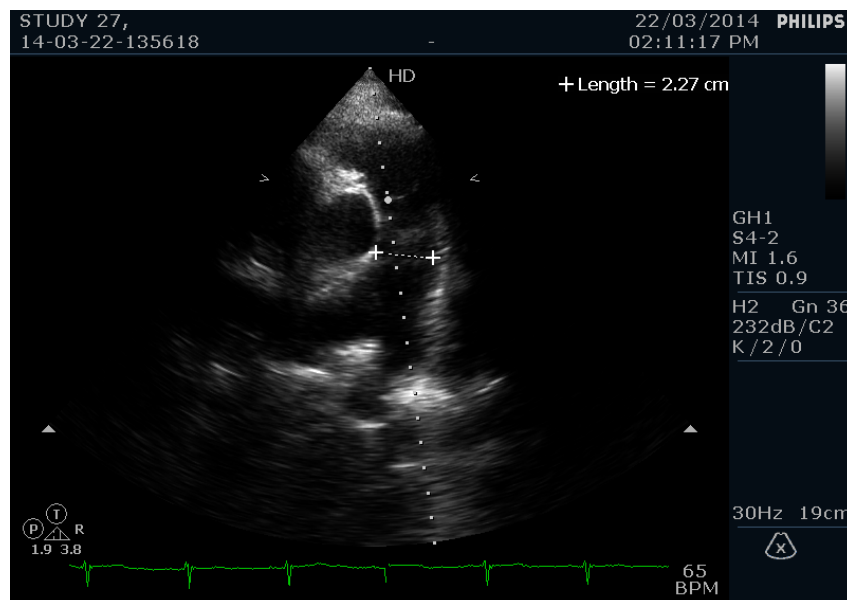


Intermediate (8mmHg) RA pressures may be downgraded to normal (3mmHg) if no secondary indicators of elevated RA pressure are present, upgraded to high if minimal collapse with deep inspiration (<35%) and secondary indicators of elevated RA pressure are present, or left at 8 mm Hg if uncertain.

*In patients who are not able to adequately perform a deep inspiration ,
an IVC that collapses $< 20\%$ with quiet inspiration suggests elevated
Right Atrial pressure.*

Pulmonary Artery Diameter

*Pulmonary artery dimension is measured between Bifurcation point and
Pulmonary valve*



All measurements were made by a single observer blinded to the patient's identity and to pre- and post-PTMC status within 48 hrs.

Percutaneous mitral commissurotomy

In our study all patients underwent PTMC by the antegrade transeptal approach using an Accura balloon and a stepwise dilatation strategy.¹⁸

The nominal balloon diameter was decided according to the height of the patient [i.e height (cm)/10 + 10 = balloon diameter].¹⁷

Echocardiography was done at the end of the procedure to assess for perforation and to look for an atrial left-to-right shunt using color flow Doppler. Successful PTMC procedure was defined as post-valvuloplasty valve area $> 1.5 \text{ cm}^2$ with no more than 2+ mitral regurgitation.

FINANCIAL SUPPORT: nil.

CONFLICT OF INTEREST: nil.

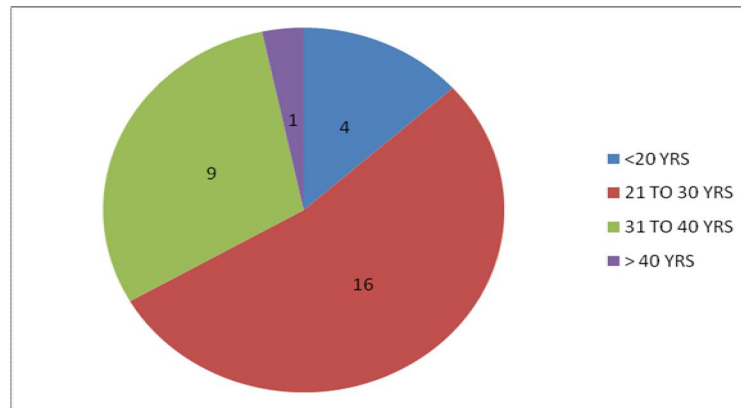
STATISTICAL ANALYSIS

Data analysis was done with use of SPSS, version 17. Descriptive statistics were used to calculate the frequency, median, mean, and standard deviation. Paired Student's t-test was used to determine the significance of differences between the various parameters before and after PTMC procedure. Unpaired Student's t-test was used to determine the differences between patients with MS and healthy subjects.

RESULTS

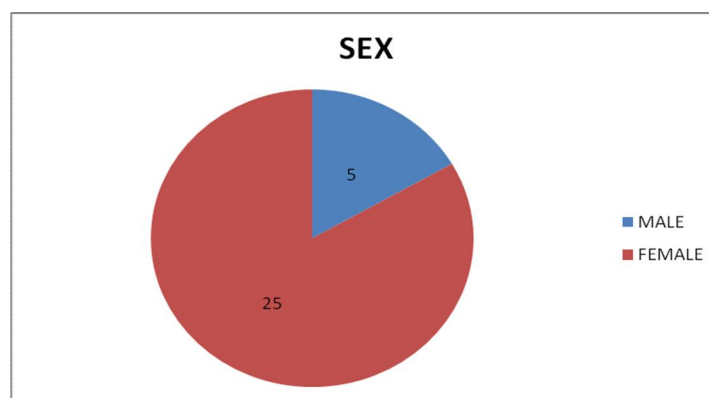
RESULTS

Age Distribution



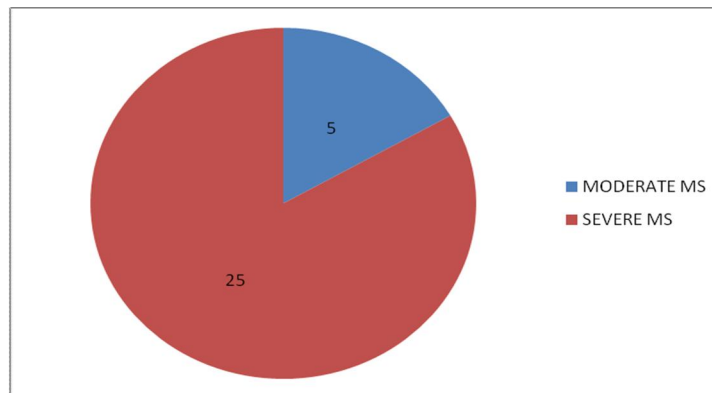
In our study there were 4 patients in the age group of <20 yrs, 16 patients between 21 to 30 yrs, 9 patients between 31 to 40 yrs and only 1 patient over 40 yrs had participated.

Sex Distribution



In our study group out of total 30 patients 25 were female patients and 5 were male patients.

MVO estimation



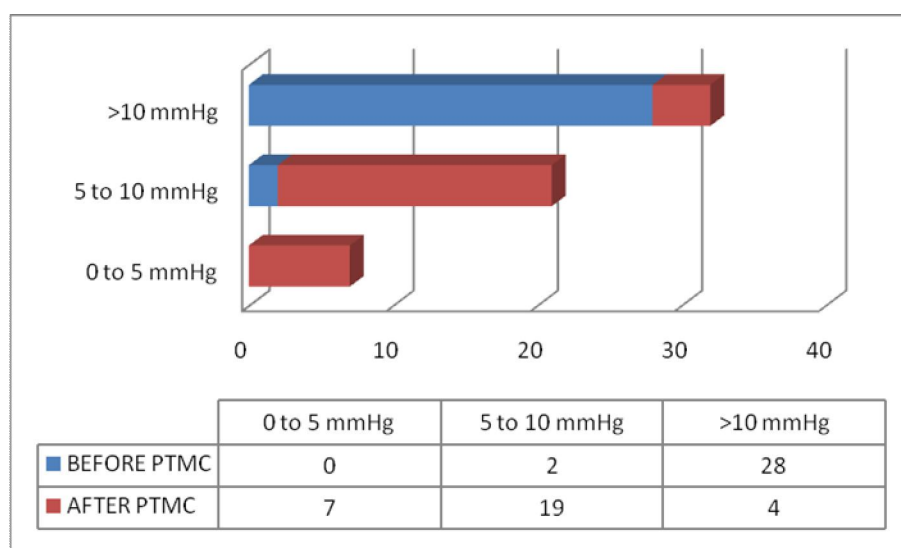
In our study out of total 30 patients 25 patients had severe MS and 5 patients had Moderate MS.

	p VALUE	Control Groups MEAN ± SD	BEFORE PTMC MEAN ± SD	AFTER PTMC MEAN ± SD	p VALUE
MVO	<0.0000001	3.98±0.617	0.86±0.17	1.58±0.17	<0.0000001

In our study 30 patients were included and MVO estimation done before and after Percutaneous Transvenous Mitral Commissurotomy within 48 hrs by planimetry . Before PTMC mean MVO area is 0.86 ± 0.17 sq cm. After PTMC mean MVO area is increased significantly to $1.58 \pm$ sq cm. After PTMC Mean MVO was increased more than 50%. This is statistically significant.

In our study control group had a mean MVO area is 3.98 ± 0.617 . In patients before PTMC mean MVO area is 0.86 ± 0.17 sq cm. MVO between control group and patients before PTMC is statistically significant.

Mean Gradient estimation



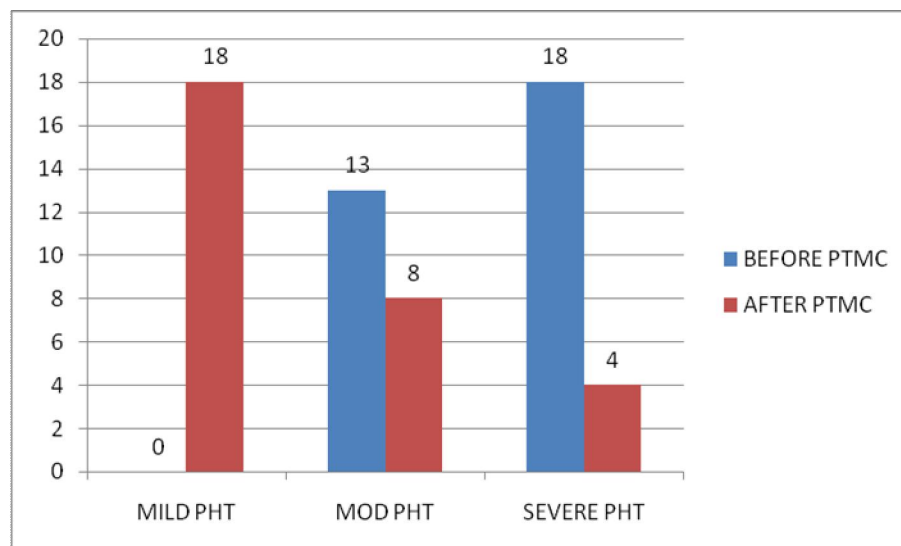
In our study before PTMC 28 patients were found to have > 10 mmHg, 2 patients had 5 to 10 mmHg but no patient have less than 5 mmHg. After PTMC mean gradient had reduced and only 4 patients had > 10 mmHg, 19 patients had 5 to 10 mmHg and 7 patients had less than 5 mmHg.

	pVALUE	CONTROL Groups	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
MITRAL Mean Gradient	<0.0000001	2.13 \pm 0.78	14.3 \pm 2.81	7.07 \pm 2.47	<0.0000001

In our study 30 patients were included and Mean gradient estimation was done before and after within 48 hrs of Percutaneous Transvenous Mitral Commissurotomy by using continuous Doppler. Before PTMC mean Mean gradient was 14.3 ± 2.81 mmHg. But after PTMC mean gradient had decreased significantly to 7.07 ± 2.47 . After PTMC Mean gradient was decreased more than 50%. This is statistically significant.

In our study the control group had an average MG is 2.13 ± 0.78 . In patients before PTMC the average MG is 14.3 ± 2.8 . Mean gradient between control groups and patients before PTMC is statistically significant.

Pulmonary Artery Systolic pressure



	p VALUE	Control Groups	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
PASP	<0.0000001	29.3 \pm 2.49	61.9 \pm 11.4	45.24 \pm 8.59	0.0000001

In our study Pulmonary Artery Systolic pressure estimation was done before and after Percutaneous Transvenous Mitral Commissurotomy by using TR velocity with continuous Doppler. Before PTMC the average Pulmonary Artery Systolic pressure was 61.9 \pm 11.4 mmHg. After PTMC Pulmonary Artery Systolic pressure was decreased significantly to 45.24 \pm 8.59 mmHg. After PTMC an average of 16.66

mmHg Pulmonary Artery Systolic pressure was decreased. This is statistically significant.

In our study out of total 30 patients, before PTMC 13 patients had moderate PHT and 18 patients had severe PHT but no patients with mild PHT. After PTMC only 4 patients had severe PHT and 8 patients had moderate PHT and significantly 18 patients had mild PHT.

In our study, control group the average Pulmonary artery systolic pressure was found to be 29.3 ± 2.49 mmHg . In patients before PTMC the average Pulmonary artery systolic pressure was found to be 61.9 ± 11.4 mmHg .The average Pulmonary artery systolic pressure between control group and patients before PTMC is statistically significant.

Mean Pulmonary artery pressure

In our study Mean Pulmonary Artery pressure estimation was done Before and After Percutaneous Transvenous Mitral Commissurotomy by using Pulmonary artery acceleration time by pulse wave Doppler.

	p VALUE	Control Groups	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
Mean PAP	<0.0000001	17.08 \pm 2.86	33.24 \pm 6.1	25.2 \pm 6.4	0.000006023

Before PTMC the average Mean PAP was found to be 33.24 \pm 6.1 mmHg. After PTMC the Mean PAP had decreased significantly to 25.2 \pm 6.4mmHg. After PTMC, an average of 8.04 mmHg Pulmonary Artery Mean pressure had decreased. This is statistically significant.

In our study the control group had a mean Pulmonary artery pressure 17.08 \pm 2.86 mmHg . In patients Before PTMC mean Pulmonary artery pressure was 33.24 \pm 6.1mmHg . Mean Pulmonary artery pressure between control groups and patients before PTMC is statistically significant.

Pulmonary artery Diastolic pressure

In our study Pulmonary Artery Diastolic pressure was derived from Pulmonary artery systolic and mean pressure using the following formula.

$$\text{PADP} = (3\text{Mean PAP} - \text{PASP})/2$$

	p VALUE	CONTROL GROUPS	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
PADP	0.00003839	10.97 \pm 3.97	19.2 \pm 4.7	14.8 \pm 3.9	0.0002171

Before PTMC the average Pulmonary Artery Diastolic pressure was found to be 19.2 \pm 4.7 mmHg. After PTMC the Pulmonary Artery Diastolic pressure had decreased significantly to 14.8 \pm 3.9 mmHg. After PTMC an average of 4.4 mmHg Pulmonary Artery diastolic pressure had decreased. This is statistically significant.

Pulmonary artery acceleration time

In our study Mean Pulmonary Artery acceleration time was estimated before and after Percutaneous Transvenous Mitral Commissurotomy by using pulse wave doppler.

	p VALUE	CONTROL GROUPS	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
PAAT	<0.0000001	135.4 \pm 8.8	92 \pm 9.8	107 \pm 16.1	0.00005413

Before PTMC the average Pulmonary Artery acceleration time was found to be 92 \pm 9.8 msec . After PTMC the Pulmonary Artery acceleration time had increased significantly to 107 \pm 16.1. After PTMC an average of 15 msec Pulmonary artery acceleration time has increased. This is statistically significant.

In our study control group had a mean Pulmonary artery acceleration time of 135.4 \pm 8.8 msec . In patients before PTMC the mean Pulmonary artery acceleration time was 92 \pm 9.8 msec . Pulmonary artery acceleration time between control groups and patients before PTMC is statistically significant.

Pulmonary artery Diameter

	p VALUE	CONTROL GROUPS	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
Pulmonary artery diameter	0.0002000	2.05 \pm 0.19	2.81 \pm 0.39	2.45 \pm 0.41	0.0009453

Before PTMC the average Pulmonary Artery Diameter was found to be 2.81 \pm 0.39 cm. After PTMC Pulmonary Artery Diameter is decreased significantly to 2.45 \pm 0.41 cm. After PTMC average 3.6 mm Pulmonary artery Diameter is decreased. This is statistically significant.

In our study control group had a mean Pulmonary artery Diameter of 2.05 \pm 0.19. In patients before PTMC a mean Pulmonary artery Diameter was 2.81 \pm 0.39 cm. Pulmonary artery Diameter between control groups and patients before PTMC is statistically significant.

Pulmonary vascular Resistance

	p VALUE	CONTROL GROUPS	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
Pulmonary vascular Resistance	0.000005112	1.45 \pm 0.30	2.19 \pm 0.31	1.70 \pm 0.28	<0.00001

Before PTMC the average Pulmonary Artery vascular Resistance was 2.19 \pm 0.31 woods unit. After PTMC the Pulmonary Artery vascular Resistance had decreased significantly to 1.70 \pm 0.28 woods unit . After PTMC an average of 0.42 woods unit Pulmonary artery vascular Resistance had decreased. This is statistically significant.

In our study ,control group subjects the mean Pulmonary vascular Resistance was 2.05 \pm 0.19 woods unit. In patients before PTMC mean Pulmonary vascular Resistance was 2.45 \pm 0.41 woods unit. PVR between control groups and patients before PTMC is statistically significant.

Pulmonary artery parameters between Nil to mild MR and Moderate MR patients

	NIL TO MILD MR			MODERATE MR		
	BEFORE PTMC	AFTER PTMC	p VALUE	BEFORE PTMC	AFTER PTMC	p VALUE
Average PASP	61.34	44.16	0.0000001	60.46	61	0.54
Average Mean PAP	32.83	24.17	0.000003	37	34.3	0.013
Average PADP	18.8	14.05	0.00010	22.2	21.1	0.204
PVR	2.19	1.63	0.0000001	2.17	2.01	0.63

Patients with nil, trivial and mild MR who undergone PTMC, after the PTMC the MR status remain the same , then there is found to have significant reductions in PASP, Mean PAP,PADP,PVR. But in patients have Nil, Mild MR before PTMC if post PTMC they develop moderate MR as a complication then there is no significant reduction in the parameters of PASP, PADP and PVR.

DISCUSSION

DISCUSSION

MVO Analysis

	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
MVO	0.86 \pm 0.17	1.58 \pm 0.17	<0.0000001

In our study 30 patients were included and MVO estimation done before and after Percutaneous Transvenous Mitral Commissurotomy within 48 hrs by planimetry . Before PTMC mean MVO area is 0.86 ± 0.17 sq cm. After PTMC mean MVO area is increased significantly to $1.58 \pm$ sq cm. After PTMC Mean MVO was increased more than 50%. This is statistically significant.

Abdenasser Drighil¹ et al study showed MVO increased from 0.91 ± 0.29 to 1.86 ± 0.43 .

Ahmad Noor et ⁴ al study showed MVO increased from 0.88 ± 0.13 cm² to 1.79 ± 0.28 cm².

Mean Gradient Analysis

	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
MITRAL Mean Gradient	14.3 \pm 2.81	7.07 \pm 2.47	<0.0000001

In our study 30 patients were included and Mean gradient estimation was done before and after within 48 hrs of Percutaneous Transvenous Mitral Commissurotomy by using continuous Doppler. Before PTMC mean Mean gradient was 14.3 \pm 2.81mmHg. But after PTMC mean gradient had decreased significantly to 7.07 \pm 2.47. After PTMC Mean gradient was decreased more than 50%. This is statistically significant.

Abdenasser Drighil¹ et al study showed Mean transmitral gradient (mmHg) decreased from 16.4 \pm 8. to 8.5 \pm 1.5 mmHg.

Ahmad Noor et al⁴ study showed Mean pressure gradient across mitral valve decreased from 18.42 \pm 7.2 mmHg to 11.85 \pm 3.6 mmHg.

Pulmonary Artery Systolic pressure Analysis

	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
PASP	61.9 \pm 11.4	45.24 \pm 8.59	0.0000001

In our study before PTMC the average Pulmonary Artery Systolic pressure was 61.9 \pm 11.4 mmHg. After PTMC Pulmonary Artery Systolic pressure was decreased significantly to 45.24 \pm 8.59 mmHg. After PTMC an average of 16.66 mmHg Pulmonary Artery Systolic pressure was decreased. This is statistically significant.

Abdenasser Drighil¹ et al study showed reduction of PASP from 46.4 \pm 32.1 to 29.1 \pm 13.4 mmHg after PTMC. An average of 17.3 mmHg PASP had decreased. In these patients MVO increased from 0.91 \pm 0.29 to 1.86 \pm 0.43. Mean transmitral gradient (mmHg) decreased from 16.4 \pm 8. to 8.5 \pm 1.5 mmHg.

In Ahmad Noor⁴ et al study PASP decreased from 60.12 \pm 17.9 mmHg to 33.4 \pm 11.9 (mmHg) p value < 0.01.

Fawzy et al² study showed Systolic pulmonary artery pressure reduction from 48.3 \pm 17.4 to 40 \pm 13.7 mmHg after PTMC.

Analysing with various studies our study also has similar reduction of PASP after PTMC

In our study total 30 patients 13 patients have moderate PHT and 18 patients have severe PHT and 0 patients in mild PHT Before PTMC . After PTMC only 4 patients have severe PHT and 8 patients have moderate PHT and significantly 18 patients have mild PHT.

After successful PTMC patients with severe PHT become moderate PHT, patients with moderate PHT become mild PHT.

Mean Pulmonary artery pressure Analysis

	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
Mean PAP	33.24 \pm 6.1	25.2 \pm 6.4	0.000006023

Before PTMC the average Pulmonary Artery Mean pressure was 33.24 \pm 6.1 mmHg. After PTMC the Pulmonary Artery Mean pressure had decreased significantly to 25.2 \pm 6.4mmHg. After PTMC an average of 8.04 mmHg Pulmonary Artery Mean pressure had decreased.

*Kamal Saad Mansour et al*³ study showed Mean PAP 38.12 ± 8.43 mmHg before PTMC that decreased immediately after PTMC to a mean value of 32.78 ± 7.89 mmHg. After PTMC an average of 5.34 mmHg Pulmonary Artery Mean pressure had decreased.

Comparing with previous studies our study also showed higher reduction of Mean PAP after PTMC.

Pulmonary artery Diastolic pressure Analysis

	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
PADP	19.2 ± 4.7	14.8 ± 3.9	0.0002171

Before PTMC the average Pulmonary Artery Diastolic pressure was 19.2 ± 4.7 mmHg. After PTMC the Pulmonary Artery Diastolic pressure had decreased significantly to 14.8 ± 3.9 mmHg. After PTMC an average of 4.4 mmHg Pulmonary Artery diastolic pressure had decreased.

Pulmonary artery acceleration time Analysis

	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
PAAT	92 \pm 9.8	107 \pm 16.1	0.00005413

Before PTMC the average Pulmonary Artery acceleration time was 92 \pm 9.8 msec . After PTMC the Pulmonary Artery acceleration time had increased significantly to 107 \pm 16.1. After PTMC an average of 15 msec Pulmonary artery acceleration time has increased.

*Kamal Saad Mansour*³ et al study showed Pulmonary artery acceleration time before PTMC with mean value of 83.78 \pm 16.87m.sec. which immediately after PTMC increased to mean value of 94.42 \pm 15.76 m.sec. After PTMC an average of 10 msec Pulmonary artery acceleration time had increased.

In our study results also comparable to previous studies.

Pulmonary artery Diameter Analysis

	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p VALUE
Pulmonary artery diameter	2.81 \pm 0.39	2.45 \pm 0.41	0.0009453

Before PTMC the average Pulmonary Artery Diameter was 2.81 \pm 0.39 cm. After PTMC the Pulmonary Artery Diameter had decreased significantly to 2.45 \pm 0.412. After PTMC an average of 3.6 mm Pulmonary artery Diameter was decreased.

In Rheumatic MS because of PHT pulmonary artery diameter was increased. After PTMC with reduction of PHT, pulmonary artery diameter also reduced significantly.

Pulmonary vascular Resistance Analysis

	BEFORE PTMC MEAN \pm SD	AFTER PTMC MEAN \pm SD	p value
Pulmonary vascular Resistance	2.19 ± 0.31	1.70 ± 0.28	<0.0000001

Before PTMC the average Pulmonary Artery vascular Resistance was 2.19 ± 0.31 woods unit. After PTMC the Pulmonary Artery vascular Resistance had decreased significantly to 1.70 ± 0.28 . After PTMC an average 0.42 woods unit Pulmonary artery vascular Resistance had decreased.

*Kamal Saad Mansour*³ et al study showed that ,PVR before PTMC with mean value of 2.25 ± 0.66 woods unit decreased to mean value of 1.76 ± 0.52 wu after PTMC. After PTMC an average of 0.49 woods unit Pulmonary artery vascular Resistance is decreased

Lung B, Vahanian A⁵: *Echocardiography in the patient undergoing catheter BMV*: et al study showed reduction in Pulmonary vascular resistance.

New onset of Moderate MR and Pulmonary artery Parameters

	NIL TO MILD MR		p value	MODERATE MR		p value
	BEFORE PTMC	AFTER PTMC	0.0000001	BEFORE PTMC	AFTER PTMC	
Average PASP	61.34	44.16	0.0000003	60.46	61	0.54
Average Mean PAP	32.83	24.17	0.00010	37	34.3	0.013
Average PADP	18.8	14.05	0.0000001	22.2	21.1	0.204
PVR	2.19	1.63		2.17	2.01	0.63

In our study patients initially presented with Nil and Trivial MR after PTMC if patients develops mild MR a complication of PTMC, In spite of New onset of Mild MR these patients showed significant reduction of pulmonary artery parameters.

In our study patients presented with nil, Trivial , Mild Mitral regurgitation showed significant reduction of PASP ,Mean PAP, PADP and PVR after successful PTMC . But patients presented with Moderate MR after PTMC as a complication of procedure that patients pulmonary artery parameters does not reduced significantly in spite of successful PTMC.

CONCLUSION

CONCLUSION

1. PTMC is a safe and effective procedure in Rheumatic mitral stenosis. After successful PTMC, pulmonary artery hemodynamics such as PASP, Mean PAP, PADP, Pulmonary vascular resistance as well as Pulmonary artery diameter was significantly reduced within 48 hrs. This is easily measured with simple non invasive echocardiographic assessment.
2. With significant reduction of Pulmonary artery pressure after PTMC the Pulmonary artery acceleration time was significantly increased.
3. If patient develop moderate MR following PTMC as a complication of procedure, Pulmonary artery hemodynamic parameters were not significantly reduced within immediate 48 hrs.

LIMITATIONS OF STUDY

LIMITATIONS OF STUDY

- 1.This study included 30 patients only.
- 2.This study focused only as the immediate impact of pulmonary artery parameters after PTMC. The Long term outcome were not evaluated.
- 3.We did not confirm with invasive Pulmonary artery hemodynamic values .

APPENDIX

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ACRONYMS

PTMC - Percutaneous Transvenous Mitral *Commissurotomy*

MVO - Mitral Valve Orifice

TR - Tricuspid Regurgitation

NYHA - New York Heart Association

PVR - Pulmonary vascular Resistance

PASP - *Pulmonary Artery Systolic pressure*

PADP - *Pulmonary Artery Diastolic pressure*

Mean PAP- Mean *Pulmonary Artery pressure*

MS- Mitral stenosis

RHD - Rheumatic Heart Disease

PHT- Pulmonary Hypertension

MR - Mitral Regurgitation

AT - Acceleration Time

MVA - Mitral Valve Area

ACRONYMS

IVC - Inferior Vena Cava

LV - Left Ventricle

RA - Right Atrium

BMV - Balloon Mitral Valvotomy

MG - Mean Gradient

AF - Atrial Fibrillation

RV - Right Ventricle

PROFORMA

Echocardiographic Assessment of Pulmonary Artery Parameters Before and After PTMC in Rheumatic Mitral Stenosis

Name Height

Age Weight

Sex

Address

Diagnosis

Balloon Size

Pre PTMC

After PTMC

IMMEDIATE

M mode

Pulmonary artery Diameter

RVOT Diameter

A wave

Mid systolic notching

Continuous Doppler

TR velocity

TR PG

Pulmonary Artery Systolic Pressure

Pul Regurgitation Peak Diastolic gradient

Pul Regurgitation End Diastolic gradient

Pul Regurgitation Diastolic pressure

Pul Regurgitation Mean Pressure

Pulse Doppler

Pulmonary Flow Acceleration time

Mid systolic notch

Right Atrial Pressure Estimation

IVC Diameter End Expiratory

Sniff Diameter

Pulmonary artery Resistance

TR velocity

RVOT VTI

$PVR = TR \text{ velocity} / RVOT \text{ VTI} \times 10 + 0.16$

Pulmonary Blood Flow

RVOT VTI

RVOT Diameter

Area

RV Stroke Volume

Heart rate

Q p

Mitral stenosis

MVO (Planimetry)

Mean Gradient

Mitral Regurgitation

Associated Lesions

MASTER CHART - STUDY GROUP

S. NO	AGE	SEX	PASP (B)	PASP (A)	Mean PAP (B)	Mean PAP (A)	PADP (B)	PADP (A)	PVR (B)	PVR (A)
1	24	FEMALE	65	39	27.83	9.25	9	8.2	1.95	1.33
2	23	FEMALE	66.3	44.8	36.3	29.6	22.1	22	2.6	1.3
3	17	MALE	81	47.1	43.1	26	24.1	15.5	1.89	1.88
4	31	FEMALE	66.3	41.6	33	20.7	16.2	10.25	1.56	1.29
5	27	FEMALE	56	50	33.4	26.8	22.1	15.2	2.59	1.71
6	24	FEMALE	70.5	63.2	37.5	33.86	21	19.2	1.81	1.49
7	18	MALE	65.3	60	36.3	33.4	21.8	20.1	1.86	1.49
8	29	FEMALE	48.2	40.1	28.2	22.9	18.2	14.2	2.4	1.9
9	25	MALE	93	52	46.6	34.1	27.6	15.4	2.47	2.1
10	25	FEMALE	53	34	30.7	21	19.5	14.5	2.4	1.7
11	30	FEMALE	69	41.6	34	21	17.2	11.2	1.6	1.3
12	23	FEMALE	75	46	39	26	21	16	1.92	1.6
13	28	FEMALE	62.4	44.8	35.1	27.1	21.4	18.2	2.4	1.8
14	39	FEMALE	67	42	34	23	18	13	2.2	1.41
15	23	FEMALE	58	45	33	24.4	21.2	14.1	2.6	1.72
16	33	FEMALE	66	47.2	37.5	33.86	23	15.2	1.9	1.52
17	35	FEMALE	48.2	38.2	26.8	21.7	16.1	13.4	2.3	1.7
18	33	MALE	71	53	41	27.8	26	15.2	2.49	2.2
19	25	FEMALE	46	34	23.3	18.1	12	10.2	2.2	1.7
20	22	FEMALE	49	39	27.3	21.8	16.5	13.2	2.5	1.74
21	39	FEMALE	65.2	42.2	34.1	23.8	18.6	14.6	2.3	1.7
22	38	FEMALE	52	44	31.3	25.4	21	16.2	1.92	1.6
23	40	FEMALE	58	41	31.4	22.4	18.2	13.2	2.5	1.8
24	45	FEMALE	45.2	32.2	21.2	16.2	9.2	8.2	1.8	1.5
25	30	FEMALE	74	56.2	40	34.1	23	18.2	2.4	2.1
26	17	MALE	48	34	23.3	16.7	11	8	1.9	1.5
27	18	FEMALE	67	62	37	34.6	21.8	21	2.3	2.2
28	30	FEMALE	56	43	32.8	24.7	21.2	15.6	2.3	1.8
29	33	FEMALE	47	39	24.6	20.4	13.4	11.2	2.2	1.8
30	26	FEMALE	67	61	37.7	35.1	23	22.2	2.4	2.34

MASTER CHART - STUDY GROUP

S. NO	PAAT (B)	PAAT (A)	MVO (B)	MVO (A)	MG (B)	MG (A)	MR (B)	MR(A)	PA DM (B)	PA DM (A)
1	100	155	1.2	1.7	15	10	TRIVIAL	MILD	2.54	2.6
2	87	97	0.6	1.2	13	8	TRIVIAL	MILD TO MODERATE	3.1	2.6
3	76	103	0.8	1.3	19	7.8	NIL	TRIVIAL	2.8	2.21
4	92	112	1	1.8	13.3	8.6	MILD	MILD	2.55	2.14
5	91	102	0.72	1.4	10	4.8	NIL	MILD	3.6	3.2
6	85	91	0.72	1.55	18.5	8.4	MILD	MILD	2.05	1.77
7	87	91	0.7	1.4	14	12	MILD	MOD	2.3	1.87
8	100	108	0.9	1.5	12	6	NIL	NIL	3.2	2.8
9	70	90	0.8	1.5	11	5	NIL	NIL	3.4	3.2
10	96	111	0.8	1.6	15	6	NIL	NIL	2.7	2.4
11	90	111	0.9	1.8	14	8.6	MILD	MILD	2.6	2.3
12	82	103	0.9	1.5	19	7.8	NIL	TRIVIAL	2.9	2.1
13	89	101	0.7	1.6	15	8	TRIVIAL	MILD	3.1	2.8
14	90	108	1	1.7	17	9	TRIVIAL	MILD	2.65	2.3
15	92	106	0.8	1.5	14	5.2	NIL	NIL	3.4	3.1
16	85	91	0.7	1.6	17.2	8.6	NIL	MILD	2.4	1.9
17	102	110	0.9	1.5	12	6	NIL	NIL	3	2.7
18	79	100	0.8	1.6	14	4	NIL	NIL	3.4	3.1
19	108	116	1.1	1.8	13	4	NIL	MILD	3.2	2.6
20	101	110	1	1.6	11	3.8	NIL	NIL	3.1	2.8
21	90	107	0.7	1.6	14	6	TRIVIAL	MILD	2.8	2.2
22	95	104	0.8	1.4	14	8.2	NIL	TRIVIAL	2.9	2.3
23	95	109	0.9	1.6	14.2	6.6	NIL	NIL	2.5	2.5
24	111	119	1.2	1.9	9	4	NIL	NIL	2.6	2.2
25	81	90	0.6	1.4	19	8	MILD	MILD	2.2	1.9
26	108	158	1.1	1.9	11	4.3	NIL	TRIVIAL	3	2.4
27	85	89	0.6	1.4	19	13	MILD	MOD	2.3	2.2
28	92	105	0.8	1.5	15	5.2	NIL	NIL	2.9	2.8
29	105	112	1.1	1.8	11	4.2	NIL	TRIVIAL	2.7	2.7
30	84	89	0.9	1.7	15	11	MILD	MOD	2.5	1.9

MASTER CHART - CONTROL GROUP

S. NO	AGE	SEX	PASP	Mean PAP	PADP	PVR	PAAT	PA DM	MG	MVO
1	24	FEMALE	29	16	9.5	1.1	140	2.1	2.2	3.2
2	23	FEMALE	26	16.5	11.75	1.21	138	2.3	3.4	4.3
3	32	FEMALE	28	13.3	5.95	1.34	146	2	1.2	4.6
4	25	MALE	30	15.1	7.65	1.62	142	1.9	3.2	4.2
5	27	FEMALE	28	20.5	16.75	1.42	130	2.3	1.4	3.9
6	36	FEMALE	26	16.8	12.2	1.21	118	1.7	1.3	3.1
7	33	FEMALE	34	22.3	16.45	1.37	126	2.2	1.8	4.6
8	18	MALE	30	14.2	6.3	1.29	144	2.1	2.2	4.7
9	42	FEMALE	32	16.5	8.75	2.01	138	1.9	1.8	4
10	26	FEMALE	30	19.6	14.4	1.93	132	2	2.8	3.2

ETHICAL COMMITTEE APPROVAL ORDER

INSTITUTIONAL ETHICS COMMITTEE

MADRAS MEDICAL COLLEGE, CHENNAI – 600 003.

EC Reg. No. ECR /270/Inst/TN/2013

Telephone No. 044 25305301

Fax 044 25363970

CERTIFICATE OF APPROVAL

To

Dr P.Ramachandran

Post graduate in DM Cardiology,

Department of Cardiology,

Madras Medical College, Chennai 600 003.

Dear Dr. P.Ramachandran

The Institutional Ethics Committee of Madras Medical College , reviewed and discussed your application for approval of the proposal "Echocardiographic assessment of Pulmonary Artery Parameters Before and After Percutaneous Transvenous Mitral Commissurotomy in Rheumatic Mitral Stenosis"

". NO. 10022014

The following members of the Ethical Committee were present in the meeting held on 04.02.2014 conducted at Madras Medical College, Chennai – 3.

1. Dr .G.Sivakumar , MS FICS FAIS Chairperson
2. Prof.B.Kalaiselvi , MD
Vice Principal, MMC, Ch3 Member Secretary
3. Prof.Ramadevi ,
Director i/c, Institute of Biochemistry, Chennai Member
4. Thiru .S.Govidasamy , BA., BL., Lawyer
5. Tmt .ArnoldSaulina , MA MSW Social Scientist

We approve the proposal to be conducted in its present form.

Sd / Chairman & other Members.

The Institutional Ethics Committee expects to be informed about the progress of the study , and SAE occurring in the course of the study , any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary , Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

PLAGIARISM CERTIFICATE

The Tamil Nadu Dr. M.G.R. Medic...Medical - DUE 31-Mar-2014

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Pulmonary Artery Parameters before and After Successful Percutaneous

Transvenous mitral Commissurotomy in

Rheumatic Mitral Stenosis

24

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirements for the award of the degree of

D.M. CARDIOLOGY

BRANCH II - CARDIOLOGY

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PATIENT CONSENT FORM

Study Details: Echocardiographic Assessment of Pulmonary Artery Parameters Before and After Successful Percutaneous Transcatheter Aortic Commissurotomy in Rheumatic Mitral Stenosis

Study Centre : Department of Cardiology
Madras Medical College and
Rajiv Gandhi Government General Hospital,
Chennai - 600 003.

Patient may check (✓) these boxes:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.

☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.

☐

I understand that the investigator of the clinical study, others working on his behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.

☐

I hereby give permission to undergo complete clinical examination and questionnaire.

☐

I hereby consent to participate in this study.

☐

Signature / Thumb impression:

Place:

Date:

Patient Name and Address:

Signature of Investigator:
Study (Investigator's Name):

Place:

Date:

ஆராய்ச்சி தகவல் தாள்

சென்னை அரசு பொது மருத்துவமனைக்கு வரும் ருமெடிக் மைட்ரல் வால்வு அடைப்பு உள்ள நோயாளிகளிடம் புறத்துளை மூலம் மைட்ரல் வால்வை விரிவுபடுத்தும் வெற்றிகரமான செய்முறைக்கு முன்னரும் பின்னரும் நுரையீரல் தமனியில் உண்டாகும் மாற்றங்களை செவிஉனரா ஒலி இதய கணிப்பு கருவி வாயிலாக ஆராய உள்ளோம் .

நீங்கள் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் பங்கேற்பதால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிக்கப்படாது என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியின் முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியில் இருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போதோ அல்லது ஆராய்ச்சியின் முடிவின் போதோ தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்

பங்கேற்பாளர் கையொப்பம்

தேதி: